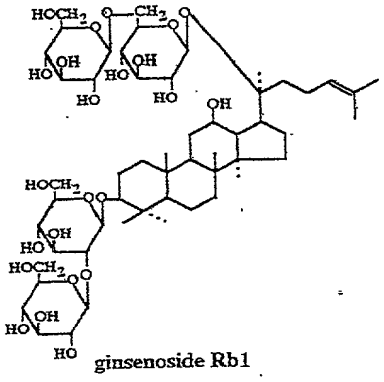
lesion) is developed in the unilateral cerebral hemisphere, nerve cell death (the secondary degeneration) is later induced in the thalamus of the same hemisphere, which keeps close synaptic connections with the cerebral infarct lesion. Subsequently, the patients deteriorate into cerebrovascular dementia depending on the progress of the unilateral thalamic atrophy or degeneration. Furthermore, if the function of the thalamus is damaged due to atrophy of the unilateral thalamus, tertiary degeneration is initiated in the other regions which have synaptic connections with the thalamus; then the cerebral functions of the patients with cerebral apoplexy may continue to deteriorate with the passage of time. In order to break the vicious circle resulting from such histological features or characteristics of the brain, drugs suppressing the above secondary neuronal degeneration are essential.

Ginsenoside $Rb_1$ is a compound having the following chemical structure (1):



ginsenoside Rb1

Ginsenoside $Rb_1$ is a known compound by a reference Shibata et al. (Shibata et al., Economic and medicinal plant research, World Scientific, Philadelphia, pp. 217-284, 1985).

Intraperitoneal administration of ginsenoside $Rb_1$ has been reported to show a tranquilizing action on the brain (Yoshimura H. et al., Eur. J. Pharmacol., 146, 291-297, 1988), but no mechanism of the action has been elucidated. In the central nervous system, the possibility has been raised that a mixture of ginsenoside $Rb_1$ and ginsenoside $Rg_1$ or ginsenoside $Rb_1$ or ginsenoside $Rg_1$ at the extracellular concentrations from $10^{-6}$ M to $10^{-7}$ M can be used for the treatment of Alzheimer's disease through activation of acetylcholine-containing neurons (US Patent No. 5,137,878: Composition and method for treatment of senile dementia). However, since it can not be said that the main cause of Alzheimer's disease is a functional disturbance of acetylcholine-containing nerve cells, this hypothesis has many problems to be solved. Moreover, the above US Patent does not address the question of whether ginsenoside $Rb_1$ can facilitate the survival of acetylcholine-containing nerve cells, namely the problem of whether ginsenoside $Rb_1$ can protect the acetylcholine-containing nerve cells or not.

The nerve cell-protective or neuroprotective action of ginsenoside $Rb_1$ has scarcely been elucidated until the studies on ginsenoside $Rb_1$ were initiated by the inventors of the present invention (Sakanaka and Tanaka). The inventors of the present

invention (Sakanaka and Tanaka) have studied, until now, on the neuroprotective action of ginsenoside $Rb_1$ using the transient forebrain ischemia model of gerbils. It has been proved that in this forebrain ischemia model, occlusion of the bilateral common carotid arteries for 3 to 5 minutes while maintaining the brain temperature at 37℃ results in a neuronal loss of the hippocampal CA1 pyramidal neurons (containing no acetylcholine) within one week after ischemia depending on the occlusion time (this event is called delayed neuronal death), and that the learning behavioral function of the ischemic animals is deteriorated (Wen T.-C. et al., Acta Neuropathol., 91, 15-22, 1996). These facts mean that the transient forebrain ischemia model of gerbils reflects the human pathologic condition of transient ischemic attack (TIA).

The one of the inventors of the present invention (Sakanaka) has proved that administering ginsenoside $Rb_1$ (10 mg/kg/day or 20 mg/kg/day: approximately 0.7 mg/day or 1.4 mg/day calculated by estimating the body weight of a gerbil at about 70 g) into the peritoneal cavity of gerbils once a day for one week in advance can significantly prevent delayed neuronal death and learning disability caused by occlusion of the common carotid arteries for 5 minutes (Wen T.-C. et al., Acta Neuropathol., 91, 15-22, 1996). However, intraperitoneal administration of ginsenoside $Rb_1$ immediately after 3- or 5-minute occlusion of the common carotid arteries showed no effect (Wen T.-C. et al.,